5-8-2021

Examining a trauma-based etiology for psychosis

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Examining a Trauma-Based Etiology for Psychosis

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A research project submitted to the Graduate Faculty of

JAMES MADISON UNIVERSITY

In

Partial Fulfillment of the Requirements

for the degree of

Educational Specialist

Department of Graduate Psychology

May 2021

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Abstract

Psychosis has been traditionally viewed through a biological lens, resulting in a narrative of the disorder that does not allow room for therapy. By applying a trauma-informed lens, psychosis can now be thought of as a trauma-based developmental disorder. This impacts how we explain the symptoms of these disorders, and how we view them overall. It also has implications for future treatment, advocacy, and research. This paper examines the bio-psycho-social symptoms of psychosis and offers trauma-informed explanations of the symptoms. Implications for Counselors are examined, as are potential limitations of this current line of research.

Introduction

Psychosis has long been considered a devastating diagnosis (Chung, Fulford, & Graham, 2007) with a poor prognosis. There continue to be many stigmas and stereotypes associated with psychotic disorders. Additionally, some disorders, such as schizophrenia, are associated with long-term disability and economic cost (Ising et.al, 2015). Of the mental health disorders, the psychotic disorders (Schizotypal Personality Disorder, Delusional Disorder, Brief Psychotic Disorder, Schizophreniform Disorder, Schizophrenia, and Schizoaffective Disorder) are one of the few that are seemingly considered incurable by the majority of practitioners in the field, a belief that likely results from a biologically based conceptualization of psychosis (Carter et.al, 2017). Despite the popularity of biological explanations of psychosis, there have been debates on how psychosis originates. How we understand the origins of a disease impacts how we conceptualize treatment. For example, if schizophrenia is derived from a brain-based disease, it becomes the responsibility of psychiatrists and medical doctors to treat, not psychotherapists. The term psychosis will include both psychotic disorders, as well as the individual psychotic symptoms such as delusions, hallucinations, disorganized thinking, disorganized motor activity (such as in catatonia), and negative symptoms such as avolition and flat affect (American Psychological Association, 2013).

With advancements in counseling around the concept of trauma-informed care, a new opportunity to reimagine psychosis is available. Authors around the globe are presenting evidence that some psychotic disorders may be the result of an extreme trauma response, rather than a biologically based disorder. Trauma has been linked with an

increased risk of hallucinations, positive psychotic symptoms, schizotypal beliefs, and first-rank symptoms (Gracie et.al, 2007). Additionally, proponents of the new diagnosis of complex trauma note that psychotic diagnoses are common for clients who could be diagnosed as having complex trauma (Everett & Gallop, 2001).

While the idea of psychosis as a trauma response may seem new; after reviewing the history of psychosis as a diagnosis, it is apparent that trauma has been associated with psychotic features for some time. Pierre Janet is one of the first clinicians to have made this link. As early as 1889, he was laying out how traumatic stress could cause symptoms that would today be considered dissociative or psychotic (Middleton et.al, 2008; Van Der Kolk, 2014) in the same year he published the first scientific account of traumatic stress (Van Der Kolk, 2014).

The current belief that psychosis is biological can be traced to Emil Kraepelin, who first formulated schizophrenia under the diagnostic name Dementia Praecox, believing it to be early-onset dementia. However, even Kraepelin later believed that the genetic components of this disease simply sensitized clients to environmental stressors, an idea in line with epigenetic theories (Jablensky, 2010). Eugen Bleuler, who coined the diagnostic name of schizophrenia, once said that schizophrenia could be an impact of an extremely negative event upon a sensitive person (Longden and Read, 2016). While the biological theory of psychosis seems to be an old one in our field, the idea of a traumatic origin of psychosis is as well. The history of our field has been a constant debate between clinicians who focus on biology, and those who focus on the effects of the environment. The history of opinions of the etiology of psychosis is no different with clinicians having theorized that psychosis and trauma are linked for over a century.

It appears that different types of adversity can have an impact on developing psychoses. Adverse childhood events, which are traumatic experiences that may negatively impact physical and brain development and functioning, have been linked with psychosis (Aas et.al, 2011; Bailey et.al, 2018; Longden & Read, 2016; Peach et.al, 2019). Some forms of childhood trauma linked with psychosis include physical abuse, sexual abuse, dysfunctional parenting, bullying, having a poor relationship with parents, growing up in institutional settings, parental death, parental separation, and neglect (Longden & Read, 2016). Those who have experienced childhood trauma are between 2 to 4 times more likely to develop psychotic symptoms (Longden & Read, 2016; Morgan & Gayer-Anderson, 2016). This increase in risk occurs even when researchers control for variables such as ethnicity, experiencing discrimination, age, substance use, other psychiatric illnesses, or family history of psychosis (Longden & Read, 2016; Morgan & Gayer-Anderson, 2016). Children who have grown up in poverty are significantly more likely to experience psychosis, even when a family history of mental illness is controlled for (Read et.al, 2009). Longden and Read (2016) noted that this "association has demonstrated a dose-response relationship; that is, the likelihood of psychosis increases relative to the extent of adversity exposure" (pg. 8). The overarching message from the research is clear: not only does exposure to childhood trauma increase the risk of psychosis, but the more trauma someone experiences, the more risk they have.

Trauma experienced as an adult can also impact the likelihood of psychosis. Traumas that have been linked to an increased likelihood of psychosis include sexual assault, trauma from war, being discriminated against, experiencing torture, witnessing domestic violence (Longden and Read, 2016). Even poverty has been linked with an

increased likelihood of psychosis (Read et.al, 2009). Factors of diversity may be closely aligned with a psychotic diagnosis. For example, schizophrenia may be more readily diagnosed in Black males. A Black male is ten times more likely than a White male to be diagnosed with schizophrenia even when they present with the same symptoms (King, 2007). There is some debate whether there is implicit discrimination in diagnosing psychotic disorders with Individuals of Color being diagnosed with more severe mental health diagnoses. One study found that Black men were diagnosed with schizophrenia, even when they only showed symptoms of Major Depressive Disorder (Kawaii-Bogue et.al, 2017).

This paper will provide an overview of the empirical literature on psychotic symptoms and disorders to examine the evidence that psychosis could be an extreme trauma response, rather than just the impact of negative biology. The symptoms of psychosis will be examined using a bio-psycho-social framework with evidence of how trauma could cause the impacts seen in individuals with psychosis. Recommendations for counselors will be provided.

Traditional Arguments for a Biological Model of Psychosis

The biological model of psychosis has traditionally dominated the field of psychiatry and therapy. However, despite years of research in this modality, the biological model of schizophrenia remains an unproven hypothesis (Jablensky, 2010). Heredity studies, combined with the neurological abnormalities found in individuals with psychosis, are considered evidence of a biological disorder, however, there are shortcomings in our understanding of both. Classic heritability studies are plagued by

methodological issues, while our understanding of the impact of trauma on the brain offers a new explanation of the neurological changes found in clients with psychosis.

Heritability

In considering biological arguments for psychotic disorders (particularly schizophrenia) it becomes important to consider the common belief that these disorders have shown high genetic heritability. This belief is the most commonly cited evidence of a biological model of schizophrenia (Fosse et.al, 2016). However, this assumption has been questioned recently by multiple authors (Fosse et.al, 2016; Longden and Read, 2016). Current studies have been unable to demonstrate the causal roles of specific genes, or combination of genes, in causing schizophrenia (Jablensky, 2010; Van Der Kolk, 2014). Additionally, the twin and adoption studies on which much of the argument is based have been criticized as methodologically flawed (Joseph, 2005).

Twin studies are considered one of the most important pieces of evidence for the heritability of schizophrenia, but there are flaws in the methodology that were used in these studies (Fosse et.al, 2016). Twin studies rely on the Equal Environment Assumption (EEA) (Fosse et.al, 2016). This is the idea that identical twins and fraternal twins are similar enough in their environment that any differences between the twin groups can be attributed to genetics. As such, it becomes important to control for environmental factors that may be different between the two twin groups. However, twin studies have traditionally failed to account for early childhood adversity and did not for them (Fosse et.al, 2016). Since we now know how closely trauma and psychosis are linked, researchers have examined this to see if trauma is an important environmental

factor to control. At least one study has found that identical twins are more similar in experiences of childhood adversity than fraternal twins, which violates the equal environments assumption for schizophrenia (Fosse et.al, 2016). As twin studies rely on this assumption for their validity, this calls into question the validity of twin studies.

When this Equal Environment Assumption fails, some researchers turn to argue that genetics is still involved. They argue that differences between the groups in trauma experience are attributable to the genes of the identical twins evoking environmental conditions not accounted for by the EEA more than the genes of the fraternal twins (Fosse et.al, 2016). This creates a circular argument, in that the reason that the argument for a genetic basis is invalid is because of the genetics themselves, which are used to revalidate the original genetic findings. However, there is another problem with this argument. When discussing early childhood adversity, this means arguing that children's genetic make-up causes them to provoke their abuse. This amounts to a biological version of victim-blaming (Fosse et. al, 2016). In one study, children's genes were cited as the reason that they experienced bullying (Fosse et.al, 2016). These arguments, aimed at avoiding issues with the EEA, not only rely on fallacious reasoning but also blame children's abuse on their genetics.

Another piece of evidence cited for the heritability of schizophrenia is adoption studies, which also had methodological problems. Some of these problems include bias towards a biological basis of schizophrenia, failure to find significance in some adoption studies, not examining the homes that the adoptees were placed in, and issues in how adoptee and relatives' diagnoses were assessed (Fosse et.al, 2016). Another issue was the prevailing belief in eugenics at the time the studies were conducted. Children who had a

family history of psychiatric illness were considered to be inferior and would have been placed into worse adoptive families (Fosse et.al, 2016). In fact, in what is considered one of the best adoption studies to date, psychosis was linked with the dysfunctional family environment of the adoptive family, not with the mental health of the birth family (Fosse et.al, 2016).

Neurological Abnormalities

There are multiple neurological abnormalities found in the brains of clients with long-term psychosis. Many of these changes are also found in individuals who experienced trauma though.

A major brain system involved in regulating emotion and responding to stressors is the Hypothalamus-Pituitary-Adrenal (HPA) axis, and this area is one of those impacted in clients with psychosis (Longden and Read, 2016). This system is believed to be triggered and sensitized in patients with psychosis by exposure to childhood trauma (Miskovic et.al, 2010; Morgan & Gayer-Anderson, 2016). In patients with Major Depressive Disorder who have also experienced childhood trauma, the HPA axis has also been found to be overactive (Zhou et.al, 2018; Dusi et.al, 2015). It is also known to be overactive in Post Traumatic Stress Disorder (Chu, 2011; De Bellis et.al, 1999; De Bellis et.al, 2010; Ford et.al, 2015; Paquola et.al, 2016). The overactivity of the HPA axis in trauma disorders and in those exposed to childhood trauma raises the question of whether this activation, when seen in clients with psychosis, is also caused by trauma. It is also an important change, as an overactive HPA axis causes a myriad of changes throughout the brain. When the HPA axis is overactive, the brain is more reactive to the environment,

causing an increased release of stress hormones such as cortisol, which is hypothesized to have developmental effects throughout the brain.

Hippocampal damage is another change in the brain common to psychosis and schizophrenia (Longden and Read, 2016). Damage to the hippocampus has also been linked to childhood trauma (Chu, 2011; De Bellis et.al, 1999; Miskovic et.al, 2010; Morgan and Gayer-Anderson, 2016), populations with PTSD (Chu, 2011; De Bellis et.al, 1999; De Bellis et.al, 2010; Ford et.al, 2015), and in those with complex trauma (Everett and Gallup, 2001). Additionally, social stressors have also been shown to cause hippocampal atrophy in monkeys (De Bellis et.al, 1999). The damage to the hippocampus is hypothesized to be due to increased amounts of glucocorticoids in the brain, which could be the result of an overactive HPA axis (De Bellis et.al, 1999; De Bellis et.al, 2010). It is important to note that while many studies have found hippocampal changes in response to trauma, a minority of studies have not found significant changes. That said, hippocampal damage offers another example of how the changes we have seen in the brain of those with chronic psychosis could be the result of a long-term sensitivity triggered by trauma.

Ventricular enlargements are one of the most commonly reported brain changes in clients with schizophrenia (DeBellis et. al, 1999; Gaser et.al, 2004; Longden and Read, 2016), and are one of the first observed (Gaser et.al, 2004). However, this symptom is not limited to just psychotic disorders. Zhao and colleagues (2018) found that in clients with Major Depressive Disorder, those with traumatic events in their childhood had enlargements of the ventricles. De Bellis and colleagues (1999) also noted this change in clients diagnosed with PTSD who suffered adverse childhood events. Additionally, it's

been found that the worse the traumatic childhood experience was (or the longer the trauma lasted), the worse the enlargement was (De Bellis et. al, 1999; Zhou et.al, 2018). These authors note that this could be a neurological adaptation for a defense to potentially dangerous situations (Zhou et.al, 2018). Additionally, ventricular size is related to neglect by parents (Zhou et.al, 2018). Ventricular enlargement offers yet another area in which the neurological changes found in those with psychosis are also present in clients with a history of childhood abuse, neglect, or trauma.

Dopamine and serotonin irregularities are often present in those with schizophrenia. These neurotransmitters are also known to be affected in other disorders, such as depression, anxiety (Wyrwoll & Holmes, 2012), and PTSD (Chu, 2011; Ford et.al, 2015; Van Der Kolk, 2014). Studies have suggested that serotonin irregularities are also present in clients diagnosed with PTSD (Ford et.al, 2015; Kelmendi et.al, 2016). Dopamine irregularities are also found in the brains of those with PTSD (Ford et.al, 2015). Additionally, studies are beginning to implicate childhood adversity with increased dopamine levels (Longden and Read, 2016; Morgan and Gayer-Anderson, 2016). Dopamine issues have been linked to neglect previously in animals (Miskovic et.al, 2010). These irregularities could be explained by an overactive HPA axis. If the HPA axis is overactive, the brain would have excess corticoids, which has been associated with problems with both serotonin and dopamine (Wyrwoll & Holmes, 2012). This offers a trauma-informed explanation of the increased dopamine found in psychosis and schizophrenia.

Atrophy of areas in the cerebral cortex is also commonly reported in the brains of schizophrenics and is also common in those who have experienced trauma (Longden and

Read, 2016). Childhood trauma is associated with diminished gray matter, especially in the frontal lobes (Carrion et.al, 2001; Longden and Read, 2016; Paquola et.al, 2016). The areas affected include the medial prefrontal, dorsolateral, orbitofrontal regions, as well as the anterior cingulate (Ford et.al, 2015; Longden and Read, 2016) which are areas also found to be impacted in clients who have psychosis (Longden and Read, 2016). A limitation of this is that brain scans of those with trauma have had mixed findings, with many studies finding evidence of cerebral atrophy, and some studies finding no difference. This could be due to issues with the state of the field in regards to how trauma is measured and diagnosed.

Reversed cerebral asymmetry is another common finding in the brains of individuals with schizophrenia (Petty, 1999), schizoaffective disorder (Riete et.al,1999), and other psychotic disorders (Riete et.al, 1999). This is also found in some clients with PTSD. Those with PTSD exhibit a variety of cerebral asymmetries (Carrion et.al, 2001; Zach et.al, 2016). Differences in cerebral asymmetry are possibly an evolutionary adaptive response to environmental stressors (Zach et.al, 2016). This is theorized to be caused by overactivity of the HPA-axis and could be a response to trauma (Zach et.al, 2016). Cerebral asymmetries, present in both PTSD and psychosis, is a potential evolutionarily adaptive response to stress that links these diagnoses.

From Genetic to Neurodevelopmental

While there is biology involved in the mental processes of psychosis and schizophrenia, a genetic basis has not been found despite decades of focused research (Read, et al, 2009; Longden & Read, 2016). A genetic explanation is not necessary though. As noted, the biological changes in psychotic disorders are similar to those experienced by those who have experienced trauma. As such, psychosis could be a developmental issue resulting from the brain adapting to trauma (Miskovic et.al, 2010). Our brain responds to environmental stressors, including trauma. Traditional theories of psychosis make the mistake of assuming that brain changes in psychotic disorders are the origin of the disorder, rather than a symptom (Longden & Read, 2016).

However, thinking of neurological changes in psychosis as a response to trauma acknowledges that our brain responds to the environment in which we are placed. It falls in line with our knowledge that early childhood abuse and neglect alters the structures of the brain associated with regulating emotions (Miskovic et. al, 2010; Paquola et.al, 2016; Wallin, 2007). As such, psychotic disorders can be thought of as the brain responds to stressors by increasing the sensitivity of the HPA axis, creating increased levels of stress hormones in the brain, which has long term detrimental developmental effects (De Bellis et.al, 2010; Miskovic et.al, 2010; Paquola et.al, 2016; Read et. al, 2009; Scott et.al, 2007). This neurodevelopmental view has existed for some time (McGlashan, 2015). Some of these detrimental effects include reduced hippocampal volume (Carrion et.al, 2001; De Bellis et.al, 2010; Paquola et.al, 2016; Scott et.al, 2007), serotonin and catecholamine neurotransmitter irregularities (Wyrwoll and Holmes, 2012), and the

smaller amygdala volume (Paquola et.al, 2016) that are observed in patients with both psychosis and trauma disorders.

Psychological

There are many psychological manifestations of psychosis. Even with the many complex psychological symptoms, theorists have noted that the symptoms are best interpreted as meaningful attempts to cope with trauma (Longden and Read, 2016). Additionally, the psychological symptoms themselves are often found in individuals exposed to trauma.

Negative Symptoms

Negative symptoms can be thought of as deficits that are manifested across multiple areas of the client's life. These deficits can occur in cognition, language, expression of emotion, goal-oriented behavior, and movement.

Cognitive Deficits

Cognitive deficits are a central aspect of schizophrenia (Aas et.al, 2011; Jablensky, 2010) that precede the illness and remain stable throughout it (Dauvermann & Donohoe, 2019; Jablensky, 2010). These deficits are often predictive of the severity of some social symptoms that individuals with psychosis will face (Dauverman & Donohoe, 2019). These deficits could be the result of overactivity of the HPA axis (Aas et.al, 2011; Schalinski, et.al, 2018).

Cognitive deficits are thought to be linked to childhood trauma and stressors (Aas et.al, 2011; Dauverman & Donohoe, 2019). Studies have shown that both working memory and cognitive function are both impaired by abuse and neglect (Aas et.al, 2011; Dauverman & Donohoe, 2019; Everett & Gallop, 2001; Schalinski et.al, 2018; Van Der Kolk, 2006). Impairment with verbal and visual memory is also known to be linked to childhood trauma (De Bellis et.al, 2010). Trauma can also impact IQ (Aas et.al, 2011) and attention span (Van Der Kolk, 2006; Van Der Kolk, 2014). Additionally, childhood trauma in individuals with complex PTSD can present with thought disordered symptoms (Everett and Gallp, 2001). Again, symptoms found in psychosis can be explained by exposure to childhood trauma.

Flat Affect

Flattened affect is a condition common to both depression and schizophrenia (Cohen et.al, 2012) and is also common with complex trauma (Everett & Gallop, 2001; Van Der Kolk, 2014). This could be a visible expression of the freeze response (Van Der Kolk, 2014). Flatness is so associated with childhood trauma that it is a common symptom in children who could fall under the proposed diagnosis of Developmental Trauma Disorder (Van Der Kolk, 2014). Flattened affect is another symptom shared by both psychosis and trauma.

Alogia

Language disturbances are also common within psychotic disorders and are central to schizophrenia (Sumiyoshi et.al, 2005). These disturbances termed alogia, include: poverty of speech, poor content in speech, delayed verbal responses, and

blocking (Sumiyoshi et.al, 2005). Alogia is also present across other mental disorders (Cohen et.al, 2012) and one study has found that speech disturbances in schizophrenia are not different from those found in other severe mental health disorders (Cohen et.al, 2012). Issues with language use make sense through a trauma-informed lens since we know that the emotional activation that often comes with trauma inhibits the areas of our brain that are responsible for speech (Everett & Gallop, 2001; Van Der Kolk, 2014). So alogia represents yet another commonality between psychosis and trauma.

Avolition

Avolition is a common psychotic symptom in which a person does not take initiative in their daily life behaviors, causing severe impairment in a multitude of life domains, such as employment, independent living, self-care, and social relationships (Trémeau et.al, 2012). This behavior could also be linked to the experience of trauma, and a lack of engagement in significant activities was a potential symptom of PTSD listed in the DSM-IV (Chu, 2011). Everett and Gallop (2001) note that many daily life behaviors, even things as commonplace as using the bathroom, could cause sensations that serve to remind survivors of their trauma. Additionally, exposure to trauma has been linked with the avoidance of medical or dental treatment if that treatment reminds them of past trauma (Everett and Gallop, 2001). Avolition is found in both those with psychotic disorders (Tremeau et.al, 2012) and those who have experienced complex trauma (Everett and Gallop, 2001), representing another way in which the symptoms of psychosis and trauma are the same.

Catatonia

Catatonia is a distinct set of symptoms related to motor immobility, a state known as a catatonic stupor (Dhossche et.al, 2012). Clients with catatonia will stop talking and stop moving for long periods, during which time they seem to lose awareness of the world around them and not visibly notice pain (Moskowitz, 2004). Some authors argue that catatonia is a long-term form of tonic immobility, a defense mechanism found throughout the animal kingdom that acts as a last "freeze response" when an animal is close to death (Karon and VandenBos, 1994; Moskowitz, 2004). Moskowitz (2004) notes that catatonia seems to occur when a person is exposed to trauma in a condition of inescapable helplessness. This trauma-based concept of catatonia is supported by findings that trauma and abuse history can lead to pediatric catatonia (Dhossche et.al, 2012; Van Der Kolk, 2014).

There are two other forms of catatonia. One is catatonic excitement, which is characterized by agitation, excitement, echolalia, echopraxia, and negativity (Moskowitz, 2004). Moskowitz (2004) argues that this form of catatonia can also be explained as a defense against an extreme threat. Catatonic excitement could be a result of sympathetic system activation so that the individual is prepared to fight or flee (Moskowitz, 2004). The third form of catatonia is negativity, which can be viewed as extreme compliance (Moskowitz, 2004) could be conceptualized as similar to what victims of chronic abuse show. Van Der Kolk (2006) notes that in the face of helplessness and inescapable chronic abuse, it's common that people become compliant and submissive. Van Der Kolk (2006) also notes that individuals in these chronic or helpless states tend to become dysregulated physiologically, which can lead to both extremely high or extremely low levels of physiological arousal. This fits in line with the proposed origin of catatonic stupor and

excitement. So the catatonia found in psychotic disorders can be understood as a trauma response.

Positive Symptoms

Positive symptoms of psychosis can be considered as those symptoms that occur in addition to deficits. These symptoms consist of dissociation, delusions, and hallucinations. Issues with reality testing could also be construed as a positive symptom. These symptoms are also linked with the experience of trauma.

Dissociation

Dissociation is common in both trauma (Chu, 2011; Everett & Gallop, 2001; Ford et.al, 2015) and psychotic disorders. Van Der Kolk (2014) notes that "dissociation is the essence of trauma."(pg. 66) This is found in populations not diagnosed with psychotic disorders as well, such as in military veterans with PTSD (Steel, 2015). This has also been observed in individuals who have had complex trauma, and Everett and Gallop (2001) note that some individuals who could be diagnosed with complex PTSD have instead been diagnosed with schizophrenia due to the severity of their dissociations. Dissociation can be viewed as a crucial coping mechanism that allows those who have experienced trauma to control painful emotions (Everett and Gallop, 2001). Psychotic lapses from reality can also be seen this way, acting as a defense against the painful childhood trauma (Blackman, 2004). Those more likely to dissociate as a defense are also more likely to hear voices (Steel, 2015), which links this symptom to hallucinations as well.

Hallucinations

Hallucinations are another symptom shared by both PTSD (Everett & Gallop, 2001; Moskowitz et.al, 2008) and psychotic disorders (Moskowitz et.al, 2008). Linkages between hallucinations and specific traumas are an old idea in our field and date back to at least Freud (Steel, 2015). Both hallucinations and intrusion of traumatic memories are involuntary and are often charged with negative affect (Peach et.al, 2019). Studies have found that the content of hallucinations in clients with psychosis is often linked to specific abuses in the client's past (Read et.al, 2009). This calls into question whether hallucinations might be some form of intrusive memory of a traumatic event (Bailey et.al, 2018; Peach et.al, 2019; Steel, 2015).

Across multiple studies, the content of hallucinations is reported by clients as a memory of negative things that have already happened, and by a larger percentage of patients as emotionally linked to traumatic events (Steel, 2015). Multiple studies have also specifically found that hallucinations are associated with early childhood trauma (Hammersley et.al, 2003; Shevlin et.al, 2007). Childhood trauma is also associated with more severe hallucinations (Bailey et.al, 2018). The link between hallucinations and childhood trauma also occurs in other disorders, such as Bipolar Mood Disorder (Hammersley et.al, 2003). Associations with trauma have been found for all types of hallucinations, including visual and tactile hallucinations (Shevlin et.al, 2007).

This link between hallucinations and trauma could be due to the way our memory is normally encoded and stored, a process called contextual integration (Steel, 2015). It's known that trauma impacts how we can store memories because in a life or death

situation a behavioral response is more imperative to survival than a full memory (Peach et.al, 2019; Steel, 2015). Priority is given to basic processing while contextual integration of memory is decreased in these situations (Steel, 2015). This is at least a partial explanation of why so many trauma survivors have difficulty remembering their trauma (Steel, 2015).

However, if an individual has poor reality testing and is prone to magical beliefs, intrusive memories or fragments of speech could be projected outside of the body (Steel, 2015). This too has a trauma-based explanation. Difficulty attending to internal senses and perceptions is found in individuals who have experienced chronic trauma (Van Der Kolk, 2006; Van Der Kolk, 2014). Hence, an individual might think a speech fragment is coming from an external source as a sound, or that an intrusive memory is perceived as an image (Steel, 2015). Steel (2015) notes that this is supported by studies finding that individuals prone to hallucinations are also likely to have worse source monitoring. Additionally, studies have found that people suffering from hallucinations also score high on measures of intrusive symptoms of trauma (Gracie et.al, 2007). These links suggest that hallucinations could be a symptom of trauma.

Delusions

Being exposed to trauma also increases the likelihood of experiencing delusional symptoms (Peach et.al, 2019; Scott et.al, 2007). This relationship has been found to display a dose-response relationship so that more exposure to traumatic events predicted more delusional symptoms (Peach et. al, 2019; Steel et.al, 2007). Studies have even found that the content of delusions has been linked to specific abuses (Read, et.al, 2009).

Steel (2015) notes that one study found that 73 percent of individuals with persecutory delusions also had recurrent threatening imagery that was intrusive. An investigation by Peach and their colleagues (2019) suggests that delusions are formed when people try to find the meaning behind post-traumatic intrusions. Delusions related to persecution occur with complex trauma as well, with some who have experienced trauma believing that they can be seen or heard by those who hurt them, and even fearing retaliation for talking in a therapy setting (Everett and Gallop, 2001).

Delusions are inherently relational, in that it requires a therapist to claim that the belief itself is irrational. However, as Van Der Kolk (2014) points out, trauma is often so horrible that it falls in the realm of unbelievable. As such, it becomes important to try to fully understand the client's history to better understand where odd beliefs come from.

Reality Testing

Reality testing is the mark of an organized mind, and a lack of reality testing is a defining feature of psychosis (Blackman, 2004). Issues with reality testing have been linked to sexual abuse (Blackman, 2004) and also occurs in other populations with exposure to trauma, especially when someone is experiencing intense affect (Everett & Gallop, 2001). These deficiencies can also be caused by being severely overwhelmed by emotional states (Blackman, 2004; Everett & Gallop, 2001). So problems with reality testing are found in both psychosis and trauma and could be understood as an impact of the overwhelming emotions that often follow experiences of trauma.

Social

Psychotic disorders are linked with multiple issues related to the way that clients interact with their world. Schizophrenia in particular is noted to cause issues with social functioning (Kalin et.al, 2015). Some social changes, such as lack of eye contact and social withdrawal, could be viewed as evolutionary defense mechanisms designed to avoid drawing contact from predators (Moskowitz, 2004). Indeed, individuals who have experienced trauma are prone to social withdrawal (Rodriguez-Srednicki & Twaite, 2006) and have a harder time interacting naturally (Chu, 2011; Van Der Kolk, 2014). This makes sense through the lens of complex trauma, as children who have experienced abuse or neglect learn that other people are a source of danger, a worldview that renders individuals isolated (Everett & Gallop, 2001). In addition to more global social issues, there are specific areas in which clients with trauma or psychosis have social struggles.

Social Cognition

Social cognitions are cognitive activities that serve as the foundation for social interactions, and there is a large amount of evidence that this is impaired in those with psychosis (Kalin et.al, 2015; Plana et.al, 2014; Statucka and Walder, 2013). This construct is multifaceted and includes constructs such as theory of mind, social perception, perception of others' emotions, and empathy (Mazza et.al, 2012). Attributional style is also considered a part of social cognition and is defined as how people explain the causes of events (Plana et.al, 2014). Social cognition issues are also present in clients with PTSD (Mazza et.al, 2012; Plana et.al, 2014). In one instance, military police officers who experienced a terrorist attack developed severe impairments in their ability to relate to others (Mazza et.al, 2012). This lines up with thinking by Van

Der Kolk (2006) who notes that impaired self-monitoring due to trauma often causes issues with recognizing the feelings and needs of those around them.

Paranoia

Paranoia can be defined as feeling threats in the absence of threatening stimuli and is common in those with psychosis. Theorists who have experienced psychosis themselves have noted that their paranoia was intrinsically linked to anxiety, and reductions in anxiety caused reductions in paranoia (Bullimore, 2010). Bullimore (2010) also notes that his paranoia always had some aspect of reality to it. Paranoia is also present in PTSD, especially traumas that involve victimization (Freeman et.al, 2013) or that are interpersonal (Gracie et.al, 2007). Van Der Kolk (2014) notes that especially for children who have been traumatized, the world is full of threats. In one study of women who had been victims of sexual assault, the authors found that paranoia was pervasive for a long time after trauma, which they hypothesized could be related to long-term damage to trust (Freeman et.al, 2013). Clients with complex PTSD have also expressed fears that abusers can still see or hear them in the therapy setting, and that they can still be harmed for speaking out (Everett and Gallop, 2001). Paranoia represents another commonality between psychosis and trauma.

Attachment Patterns

Patients with schizophrenia and psychosis often have marked impairments with attaching with others, but this pattern of insecure and disorganized attachment also seems to be a factor in increasing the likelihood of psychosis (Read et.al, 2009). This fits with a trauma-oriented theory of psychosis, as childhood neglect and trauma harm attachment

(Bailey, 2018; Liotti & Gumley, 2008; Wallin, 2007), which has even been hypothesized as a cause of negative symptoms (Bailey, 2018). Disorganized attachment is also linked with experiences common to both trauma and psychosis, such as problems with coping, metacognition, controlling aggression, regulating emotions (Liotti & Gumley, 2008), and also with increased dissociation (Wallin, 2007). Finally, disorganized attachments cause problems attaching successfully in interpersonal relationships for those with psychosis, a problem also shared by those who have experienced trauma (Everett & Gallop, 2001; Ford et.al, 2015, Van Der Kolk, 2014). Disorganized attachments can also be an inherited trait from parents, who themselves may have been subjected to similar dysfunctional parenting which acknowledges the role of attachment in psychosis (Liotti & Gumley, 2008; Read & Bentall, 2012). This is found with trauma as well, with one study finding that children of Holocaust survivors are more likely to have a disorganized attachment style and develop PTSD in response to trauma (Van Der Kolk, 2014). Attachment issues are another symptom that is found in psychosis that may be attributable to trauma.

Discussion

As noted, there are many commonalities between symptoms of psychosis and trauma. There are no major symptoms of psychosis that cannot be found as a response to trauma. If trauma can produce all of the symptoms of psychosis, exploring a trauma-based developmental model for psychosis becomes imperative. A developmental model for psychosis, instead of the current biological one, has wide-ranging consequences for treatment, advocacy, research, and prevention for counselors.

Treatment Implications

The way we view diagnosis influences the way we practice (Carter et.al, 2017). Those who view disorders biologically tend to use medicine as a front-line treatment, while those who view psychotic disorders as psychosocial in etiology prescribe therapy (Carter et.al, 2017). By adopting an integrative approach acknowledging both nature and nurture, we can take a more balanced approach. This approach matters greatly, especially since theorists have begun to question both the safety and efficacy of antipsychotic medication (Prouty, 2009). Schwartz and Summers (2009) point out that an approach focused solely on the prescription of medication only acknowledges a biological basis for psychosis, and does not offer the possibility of complete recovery. This creates a space in which the client has no real hope to recover (Schwartz & Summers, 2009), which gives them very little reason to buy into the therapeutic alliance. Kallie Coman (2011) notes that this happened in her journey of recovery from schizophrenia and she came to believe that she believed that she could never recover or live the life she wanted.

This demonstrates much of what is wrong with the way the field approaches psychotic illnesses. Despite the seemingly widespread belief in the intractability of psychotic disorders, there is a growing body of literature describing psychosis as treatable, and even as curable. Various approaches (representing nearly the whole field of counseling) have claimed some success in treating patients with psychosis. These approaches include psychoanalytic (Karon and VandenBos, 1994), psychodynamic, cognitive (Karon and Silver, 2009), humanistic (Prouty, 2009), family systems (Karon and Silver, 2009), and more integrative (Rakitzi & Georgila, 2019) approaches. Trauma-oriented approaches such as EMDR and exposure therapies have also been effective in those with psychosis and co-morbid PTSD (Steel, 2015). Cognitive Behavioral Therapy

and Social Skills Training have been found to reduce negative symptoms (Kalin et.al, 2015). It's also been suggested that targeting traumatic memories and intrusions can reduce delusions and hallucinations (Peach et.al, 2019). At the very least, the research suggests that we should be assessing for trauma when we encounter psychosis, which is a current known shortcoming in assessments of psychosis (Peach et.al, 2019; Shevlin et.al, 2007). At the center of treatment success is the tenacity of the therapist in working to establish a sound therapeutic relationship (Karon and VandenBos, 1994; Prouty, 2009; Schwartz & Summers, 2009).

The evidence on delusions and hallucinations being linked to past traumas encourages us as clinicians to better understand a client's past before making clinical judgments. As Van Der Kolk (2006) noted about traumatic symptoms, without understanding a client's history, emotions and actions may appear "out of place" and "bizarre". At times, this may even be remnants of behavior from the time in which the event happened (Van Der Kolk, 2006). Only by understanding a client's full psychosocial history, will some symptoms become understandable. It also has implications for the way counselors should greet hallucinations and delusions. If these experiences are linked to past trauma, refusing to engage with them could be likened to greeting a trauma disclosure with disbelief. Not having disclosures believed is something that has been considered traumatic in other populations (Everett & Gallop, 2001). As such, by treating these symptoms as untrustworthy, we may be re-traumatizing the client. Indeed, those diagnosed with psychotic disorders have reported feeling dehumanized by their treatment (Bullimore, 2010; King, 2007). Everett and Gallup (2007) note that we should engage

with delusions and hallucinations as a meaningful form of communication to be engaged with.

This also suggests a change in treatment related to how we view hospitalization. Hospitalization is not uncommon for those with psychotic symptoms; however, hospitalizations can fuel paranoid ideations (Bullimore, 2010). It can also be re-victimizing, as it represents an instance in which clients are forced to do what someone in a position of power (in this case the therapist) wants of them (Everett and Gallop, 2001). This can lead to a fear of admitting positive symptoms such as hallucinations, because of the fear that doing so will lead to hospitalization (Coman, 2011). If we start to view psychosis and schizophrenia as disorders arising from fear (Karon and VandenBos, 1994) and trauma (Longden & Read, 2016) then it becomes even more important to interact with these clients in ways that do not fuel their fear.

Acknowledging a trauma-informed and neurodevelopmental explanation of psychosis does not preclude the use of counseling. As we have learned more about the brain, more evidence has accumulated that new learning can alter the structure of the brain (Mancke et. al, 2018). Patients with schizophrenia who underwent cognitive remediation therapy showed increased gray matter in the hippocampus, amygdala, and the fusiform gyrus (Mancke et. al, 2018). Dialectical Behavior Therapy has been found to increase gray matter in the anterior cingulate cortex (Mancke et. al, 2018). Changes in brain activity in the pre-frontal cortical activity have also been shown with psychodynamic and cognitive-behavioral therapies (Wiswede et.al, 2014). Programs targeting social cognition deficits in schizophrenia have also been shown to increase fMRI activation in multiple cortical lobes (Statucka and Walder, 2013). Mancke and

colleagues (2018) note that to create changes to the amygdala or hippocampus, therapy would likely have to take place over a long period. This aligns with Karon and VandenBos (1994) suggestions that psychotherapy for schizophrenia and psychosis needs to be in-depth, lasting a good amount of time, and be more pernicious than the disorders themselves. This is supported by a study finding that relationally focused psychodynamic therapy conducted over 8 months can decrease activity in the amygdala and basal ganglia (Wiswede et. al, 2014). Evidence is accumulating across the field that even if we are confronted by neurological changes, there is still the potential to undo some of the presented damage.

Prevention Implications

In terms of prevention, Read and his colleagues (2009) note that an understanding of psychosis as a response to childhood trauma necessitates more programming aimed at helping care for children's mental health. Some community programs targeted at children have been noted to decrease schizotypal traits when measured 20 years after the intervention (Raine et. al, 2003). Additionally, early intervention in the form of intensive psychotherapy for those showing early signs of psychosis has been effective in reducing symptoms years later (McGlashan, 2012). CBT interventions before the first episode of psychosis are effective in reducing symptoms and onset of psychosis and lowering long-term treatment costs (Ising et.al, 2015; Morrison et.al, 2002).

Research Implications

This model has a large range of implications for research as well. Read, Bentall, and Fosse (2009) note that despite a continued lack of findings for a genetic basis for

psychosis, research searching for a genetic basis occurs 16 times as often as research on psychosocial etiology. Schizophrenia is a complex disorder, with some theorists suggest that it is a series of different disorders, and others suggesting that psychosis should be viewed as a continuum (Chung, Fulford, and Graham, 2007; Jablensky, 2010). If at least some instances of psychosis are better explained by exposure to trauma and are treated, it could allow us to better focus on which instances of psychosis are organic. It also allows for chances to increase research on preventative programs supporting individuals who have gone through crisis and trauma.

Limitations

An important limitation of this research is that not all individuals who experience childhood trauma go on to experience psychotic symptoms (Morgan and Gayer-Anderson, 2016). As such, greater research is needed on risk and resiliency factors between psychosis and trauma. Some research suggests that this may be related to either the severity or length of abuse, with some trauma types having different long-term impacts (Evans et.al, 2013). It is also possible that social support is a buffer against the negative impacts of trauma (Evans et.al, 2013). It is also important to acknowledge that this limitation is shared by the field in regards to trauma as well since not all individuals who experience trauma experience mental illness in the aftermath (Everett & Gallop, 2001).

Another limitation is that some question whether disclosures of abuse from clients suffering from psychosis can be considered accurate. However, false allegations of childhood abuse are also rare (Everett & Gallop, 2001). Additionally, studies have shown

that disclosures from those with psychosis can be considered reliable (Longden and Read, 2016).

Additionally, we know that there are environmental factors that can cause psychotic symptoms. Some of these include heat injuries (Canuso et.al, 2008), adverse reactions to antibiotics (Mostafa and Miller, 2014), substance use (American Psychological Association, 2013), and medical conditions (American Psychological Association, 2013; Kampfe, 2014).

Morgan and Gayer-Anderson (2016) also note that a major issue with this theory is that it is new. As such, much of the associations between psychotic symptoms and childhood trauma need more research to better understand (Morgan & Gayer-Anderson, 2016). There is also a wide variability within the research of this part of the field. For example, the field of abuse research has issues with definitions of what constitutes abuse, and in whether or not respondents recognize abuse in their lives when participating in research (Everett & Gallop, 2001).

This limitation of abuse research is shared by trauma research as well. Much research within the field of trauma is focused more on PTSD. This form often takes the form of examining people who have been exposed to a traumatic event and finding differences between the group that developed PTSD, and those who did not. One problem with this methodology is that it does not adequately explore the cumulative nature of excessive stress on the structures of the brain. Further development of relational trauma diagnoses as proposed by Van Der Kolk (2014) would allow a deeper understanding of

the chronic effects of trauma and may serve to further reinforce the hypothesis that psychosis can be a developmental response to trauma.

Conclusion

The link between psychosis and childhood trauma is compelling. It offers a chance to redefine an entire area of the field around the idea of trauma-informed care. Doing so allows us greater opportunities in the realm of research, advocacy, prevention, and treatment. The recent movement in our field towards the inclusion of a complex trauma diagnosis allows a reimagining of diagnosis, with a trauma spectrum. With the growing evidence of linkages between trauma and psychosis and our knowledge of how the brain and body respond to trauma, psychotic disorders could be considered the end of that spectrum.

This model of psychosis also offers an opportunity to engage a population in a way that offers more hope than has traditionally been given. Working from a trauma-informed perspective allows us a chance to create a meaningful intersubjective space in which clients have the hope and support to heal. Additionally, it offers counselors who confront psychotic symptoms an increased toolbox of approaches to use, as trauma approaches could now be included in treatment options. Counselors intervening early with trauma-informed approaches offer a chance to decrease symptoms while decreasing the economic cost of the disorders.

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